

Sexually transmitted diseases in children: non viral including bacterial vaginosis, *Gardnerella vaginalis*, mycoplasmas, *Trichomonas vaginalis*, *Candida albicans*, scabies and pubic lice

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There are important anatomical and physiological differences between the genital organs of adults and children. In a child the vulva is located more anteriorly than the adult with the clitoris, labia minora and hymen relatively more prominent. The vagina has a shallow posterior fornix and is of comparatively greater length than in the adult. The cervix is tiny with few glands and is rarely a source of leucorrhoea. The vulva lacks the characteristic protection of subcutaneous fat and hair present in the adult. At birth the presence of maternal oestrogens produces transient vulval and hymenal engorgement with a thickened glycogen-rich epithelium and a consequent physiological leucorrhoea. The vaginal epithelium thins and the glycogen disappears; thus the introital area is normally redder in young girls because only this thin epithelium overlies the vascular stroma.¹ The vulva is delicate and sensitive, easily traumatised by physical injury and susceptible to infection.² Disruption of the epithelial integrity from whatever cause may permit colonisation or opportunistic infection by any potential pathogen. With the approach of puberty, endogenous oestrogen is produced and the epithelium thickens and contains glycogen. The pre-menarcheal cervix secretes mucus from the columnar epithelium under oestrogenic influence. These differences will influence the microbiological flora of the genital tract, and therefore the micro-organisms which can be considered potentially normal and those that are potentially pathogenic.

Normal flora

At birth, the vaginal pH is acid.³ The newborn child's vagina is sterile. Over the first 12-24 hours staphylococci, enterococci, and diphtheroids appear, but are replaced within 2-3 days by lactobacilli.⁴ As the influence of oestrogen diminishes, the flora is replaced by cocci and Gram negative rods, and the pH rises producing an alkaline environment. Lactobacilli may persist through childhood,⁵ but appear to be isolated more often from older girls⁶ as the pH decreases prepubertally.

Our current understanding of the normal vaginal flora in childhood is limited. The information has been obtained from studies with different aims and with a wide age range of children. Some have compared children with vulvovaginitis to a group of "controls" without addressing the possibility of sexual abuse.^{1 3 6-8} Other investigators have used control groups which do not exclude girls with a

history of abuse^{9 10} or were screened only for current sexual activity.¹¹ Some studies had no control groups.¹²

A high prevalence of diphtheroids and *Staphylococcus epidermidis* has been noted from several studies^{3 7} and may not represent just contamination from the skin.⁶ Common childhood pathogens include *Staph. aureus*, *Escherichia coli*, *Haemophilus influenzae*, β -haemolytic streptococci of Lancefield groups A and B, and *Streptococcus milleri*. *Staph. aureus* has been reported in 5-14% of children with vulvovaginitis^{7 13} and 0-7% of asymptomatic children^{6 13} in both abused and non-abused children. *Esch coli* has been isolated from abused and control children and has been reported in asymptomatic children^{6 7} and children with purulent vulvovaginitis.^{7 13} It has been suggested that hygiene may play an important role in colonisation with this organism as it is found predominantly in girls under two years of age.⁶ *H influenzae* has been reported in 2-4% of children with vulvovaginitis but not in asymptomatic control subjects,^{7 8} although Gerstner isolated *H influenzae* from two of 31 girls without symptoms.¹³ Infection with *H influenzae* has been found to occur simultaneously with isolation of *H influenzae* from both the nose and vagina.³ Group A streptococci have been recognised as a specific cause of vulvovaginitis in 2-10% of symptomatic children,^{2 7 8 14-16} some of whom had documentation of previous sexual abuse.¹⁵ Group B streptococci have been isolated from sexually abused children with vulvovaginitis.¹⁵ In Hammerschlag's study, of 100 healthy children,¹⁷ 20% overall were colonised with Group B streptococci. In children under 3 and those over 11 years of age, 19% and 25% respectively had anogenital colonisation but only 4% of children between 3 and 10 years ($p < 0.025$). Pharyngeal colonisation was found in 15% of girls under 11 years and 5% of those over 11 years. There is other evidence to suggest that the vaginal flora in children is related to the nasopharyngeal flora.^{3 16 17} The presence of a specific organism in the child's vagina may indicate normal commensal flora and not necessarily indicate pathogenicity.

Bacterial vaginosis and *Gardnerella vaginalis*

"Non-specific vaginitis" was the term originally used to distinguish this syndrome from the specific vaginitides associated with yeasts and *Trichomonas vaginalis*. In 1955, Gardner and Dukes characterised a syndrome in adult

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women with vaginal discharge which grew *Haemophilus vaginalis*.¹⁸ Non-specific vaginitis was then called *H vaginalis* vaginitis. The term vaginosis was introduced to indicate a lack of significant inflammation associated with the vaginal discharge.¹⁹ The term bacterial vaginosis (BV) was adopted to indicate that various bacteria rather than other organisms seemed to be associated with this syndrome. The accepted clinical diagnostic criteria for BV in adult women are the presence of three out of the following four signs.²⁰

- i) Vaginal fluid with a pH > 4.5
- ii) Homogenous adherent discharge
- iii) Fishy odour on addition of 10% potassium hydroxide (KOH) to the discharge (amine test).

iv) Presence of clue cells on microscopy.

Unfortunately, one of the four diagnostic criteria cannot be readily applied to pre-pubertal children because the immature vagina produces an alkaline environment. It is difficult to make any sense of the published literature concerning vaginosis in children because some studies have looked at isolation of *G vaginalis* and others at the syndrome "bacterial vaginosis". The detection or quantification of *G vaginalis* cannot be used as a diagnostic test for BV. Although Gardner and Dukes recovered this organism from 92% of women with bacterial vaginosis,¹⁸ other studies have isolated *G vaginalis* from women without BV and it can be a common member of the indigenous vaginal flora.²¹⁻²³

The significance of *G vaginalis* in the vaginal discharge of a pre-pubertal female is uncertain. *G vaginalis* has been cultured from various sites in new-born infants including gastric aspirates^{24,25} and oral swabs,²⁶ indicating that this organism can be transmitted vertically, but how long an infant remains colonised is unknown. Sexual abuse is another possible route of transmission.²⁷ De Jong reported a case of assault in a 12 year old girl in whom *G vaginalis* was isolated in addition to *Candida albicans*.²⁸ Bartley reported that pre-pubertal girls with a history of sexual abuse were more likely to have *G vaginalis* on vaginal culture (14.6%) than the control patients (4.2%) or patients with genitourinary complaints (4.2%).²⁹ However, the validity of the control group of patients in this and other studies is questionable. After follow-up evaluation, some of the control group were reassigned to the abused group because of disclosure of previous sexual abuse. In Hammerschlag's study 13.5% of vaginal cultures grew *G vaginalis*.⁶ The organism was more common in children aged two years and under and between 11-15 years, reaching significance in the latter group. The children were from a predominantly black, disadvantaged inner-city population and the question of sexual abuse was not specifically addressed. Frau and Alexander³⁰ stated that *G vaginalis* infection in children is most likely to be from sexual transmission, but they cited no references to support this statement. Ingram *et al*³¹ reported no significant difference in the isolation rate of *G vaginalis* between children who

had a history of sexual contact and/or were infected with gonorrhoea or *Chlamydia trachomatis*, those children who were evaluated for abuse and a "control" group who were children of friends of the authors! There was a correlation with age and race. Gardner¹⁵ isolated *G vaginalis* in 7% of 209 sexually abused girls compared to 1% of 108 controls, a finding which reached statistical significance ($p < 0.01$). *G vaginalis* has been recovered from the rectum of two (9%) of 22 sexually inexperienced children.³² The possibility of anal to vaginal transfer may account for the presence of this organism in vaginal cultures of children.

As in adults, BV may be linked to sexual activity but sexual transmission has not been clearly documented. BV should not be considered an exclusively sexually transmitted disease.³³

Girls with BV and/or *G vaginalis* can be asymptomatic.^{6,9,29,33} The diagnosis may result from evaluation of children who have been sexually abused. In Hammerschlag's study⁹, 8 of 31 abused girls had odour or clue cells or both seven days after sexual abuse, even though four had normal findings initially. Only one of 23 non-abused girls had possible non-specific vaginitis, on the basis of odour alone. Some girls may have vulvovaginitis,⁶ vaginal odour¹¹ and discharge.²⁸ It has been reported that *G vaginalis* is the second most common cause of vaginitis in sexually abused girls after gonorrhoea.^{6,14,34}

Vaginal pH is not a reliable parameter for the diagnosis of BV in children. Specimens of vaginal discharge can be obtained by using plastic loops. The amine test is best performed by mixing a drop of 10% KOH solution with the discharge. A smear can be examined microscopically for the presence of clue cells on a wet mount. Material for culture for *G vaginalis* can be obtained by swabbing the vaginal introitus with a cotton wool swab, inoculating on to human blood agar screened with nalidixic acid and polymyxin B.³⁵ However, the value of *G vaginalis* culture is questionable. Others have obtained vaginal washings by injecting 2mls^{36,37} of sterile saline into the vagina, aspirating the fluid and using this for the above tests. The sensitivity and specificity of these diagnostic methods are not well documented for prepubertal children.

In the absence of symptoms, it may not be necessary to treat BV as it may resolve spontaneously.³³ Metronidazole can be used, but with caution in young children, at a dose of 15-20 mg per kilogram per day to a maximum dose of 250 mg in three divided doses for seven days.³⁷ Other treatments include clindamycin or co-amoxiclav in the appropriate dosage for seven days.³⁸

The isolation of *G vaginalis* and/or presence of BV should alert clinicians to seek historical and behavioural indicators of possible abuse. Cultures should also be taken to exclude other sexually transmitted organisms such as *C trachomatis*, *Neisseria gonorrhoeae* and appropriate further evaluation of the child undertaken as considered necessary.

Mycoplasmas

In adults, vaginal colonisation with *Mycoplasma hominis* and *Ureaplasma urealyticum* is strongly correlated with sexual activity,³⁹ race^{11 40} and hormonal status.^{41 42} The isolation rates for *M hominis* and *U urealyticum* are reported as 10–20% and 30–75% respectively in sexually active adults. In children there are two published studies of vaginal colonisation in prepubertal children reporting a range of 6–11% for *M hominis* and 11–27% for *U urealyticum*.^{6 39} In infants under two years of age colonisation with mycoplasmas is thought to be related to vertical transmission.⁴³ A small number of male children have been found to harbour mycoplasmas in the rectum.^{32 39} In older children it is possible that these organisms may be acquired sexually. Waites *et al*⁴⁴ reported a case of a ten year old girl with vaginal discharge after sexual abuse from whom both *M hominis* and *U urealyticum* were isolated. Coury⁴⁵ reported a significantly increased rate of colonisation with *U urealyticum* (48%) amongst 40 sexually abused children than in an historical control group. Studies of normal,⁴⁶ virginal³³ and control girls^{10 27} have reported isolation rates for mycoplasmas between 6–17%. Some studies have shown a significantly higher isolation rate for *U urealyticum* and *M hominis* from abused girls compared to controls.^{15 45} Others have not shown any difference.⁴⁰ Therefore the use of *M hominis* and/or *U urealyticum* as a marker of sexual abuse in older children remains controversial.

The presence of *M hominis* and *U urealyticum* may not result in any symptoms.¹⁰ They can also be isolated from children with vaginal discharge,^{12 47 48} and have been isolated significantly more often from children with non-specific vaginitis in at least one study.¹¹

The value of identifying mycoplasmas is doubtful. They need not necessarily be pathogenic, and on the basis of the present literature the organisms may or may not be a marker for sexual abuse. The isolation of mycoplasmas is very dependent upon specimen quality and culture method. Optimal isolation rates require the use of a semiquantitative liquid medium, with transfer to a solid medium for identification.³⁹ Published studies have utilised different techniques and results may not be comparable. The reliable isolation of mycoplasmas should be carried out by arrangement with a reference laboratory.

Treatment is usually unnecessary unless the child has vaginal discharge for which no other cause is found. There is no data on treatment efficacy in children. If thought to be clinically significant, *U urealyticum* can be treated with erythromycin at a dose of 40 mg per kg per day in four doses for 7 days. The treatment of choice for *M hominis* is clindamycin.⁴⁸ The child should be reviewed to ensure satisfactory resolution of symptoms.

Trichomonas vaginalis

Donné in 1836 first described the presence of the flagellate protozoan *Trichomonas vaginalis* in purulent secretions of the genital tract in

both men and women.⁴⁹ *Trichomonas* was reported in the 1950s to be present in 10–25% of women in the reproductive years of life and in 15% of men with non-specific urethritis.⁵⁰

Trichomonal infection has been described in neonates. In the first few weeks of life under the influence of maternal oestrogens, the new-born may be susceptible to infection with *T vaginalis* transmitted vertically. Approximately 5% of female babies born to infected mothers have trichomonal infection.⁵¹ These organisms have been shown to persist for up to nine months in the absence of treatment.⁵² Sexual abuse as a mode of transmission has also been documented.^{12 52–54} Infection with *T vaginalis* had previously been reported in pre-adolescent girls but the possibility of sexual acquisition was not considered until 1978.⁵³ In studies comparing sexually abused and non-abused girls the incidence varies between 0.5% and 8%.^{6 8 9 33}

The possibility of transmission by non-sexual means cannot always be excluded. Other methods of transmission apart from sex are more likely when infection is found in premenarcheal children. McCullagh in 1953⁵⁵ thought the main cause of infection was toilet seats but could not give any evidence to support this view. However, this prompted Whittington⁵⁰ to assess the validity of the statement by checking the survival time of trichomonads in vaginal exudate on bacolite, polished wood and absorbent wood seats. Her work showed that the parasite remains viable for 45 minutes maximum. She also conducted experiments which showed that women with *T vaginalis* may leave infected material on a toilet seat. However, the question of whether *T vaginalis* can actually be acquired from a toilet seat remains unanswered. Other sources of infection have also been suggested for which there is no reliable evidence, including contaminated towels, underwear, food, water and swimming pool water. It has been suggested that transfer of intestinal trichomonads may occur from the anorectal region into the bladder and vagina⁵⁶ but this is unproven. Catterall⁵⁷ suggested that the most frequent method of transmission is by the sexual act, although infection may be acquired by other means in a small proportion of cases. In children beyond the first year of life the finding of genital infection should suggest sexual contact and warrants evaluation for sexual abuse and investigation for other sexually transmitted diseases.

Vulvovaginitis is the commonest presenting symptom in children. The discharge can be green and malodorous and is often watery in consistency.⁵⁸ Feo found that 3.6% of asymptomatic girls aged between 1–9 years had a trichomonal infection.⁵⁹ In neonates, *T vaginalis* can cause nasal discharge and otorrhoea.⁶⁰ Pneumonia has also been reported.⁶¹ *T vaginalis* has also been found in the urine of a seven year old boy without urinary symptoms.⁶²

To diagnose trichomonal infection, vaginal secretion should be obtained either with a cot-

ton wool tipped swab from the vaginal introitus or with a plastic loop if vaginal discharge is present. A fresh wet preparation should be examined microscopically, preferably using phase contrast microscopy and/or a dry smear can be stained with acridine orange fluorescence staining.³⁵ Culture medium (e.g. trichosel broth) can be inoculated with the vaginal/vulval secretion but culture is an imperfect gold standard.⁶³ The sensitivity and specificity of these various diagnostic methods have not been documented for children.

The treatment of choice is metronidazole either as a single dose of 2 g in adolescents or 7.5 mg per kg per day to a maximum of 250 mg tds over 7 days. It should be used with caution in young children. A search should be made for other sexually transmitted diseases. An infected child should have follow-up evaluation to ensure that the treatment has been successful.⁵³

Candida albicans

Reports of fungal infections in the literature before 1930 have little meaning owing to faulty identification of the organisms.⁶⁴ More recent data on the presence of *Candida albicans* in children are available from studies of the aetiology of vulvovaginitis. *C. albicans* has been found to be part of the normal vaginal flora in children.^{3,67} Vaginal colonisation with these species is related to age, being more common in adolescents⁶ presumably because of a preference for an oestrogenic vagina.² The commonest yeast isolates overall are *C. albicans*, but *C. tropicalis*, is found more often in children under two years.⁶ Primary mucocutaneous candidiasis is a common problem in the neonate and infant. The source of the infection is thought to be maternal vaginal candidiasis. Adults can acquire candidal infections through sexual contact⁶⁵ and sexual transmission in children following sexual abuse has been reported.²⁸ Association of symptomatic vaginal candidiasis with sexual abuse has not been proven and the incidence of carriage in non-abused pre-pubertal girls is unknown. De Jong recommended that *C. albicans* should not be considered as normal flora in symptomatic children and adolescents but should raise the possibility of sexual abuse. Other studies have shown no difference in isolation rates between sexually active and non-active children.¹¹

C. albicans is traditionally considered the commonest specific cause of vulvovaginitis in children.² Although Hammerschlag demonstrated that 28% of girls aged 2 months to 15 years had vaginal yeasts, only 30% had an abnormal vaginal discharge. Other studies have identified *C. albicans* as a cause of genital discharge, irritation, pain and redness.^{8,66} As in adults, candidal vulvitis has a spectrum of severity from mild erythema with pruritis to an intense red, macerated, weeping excematoid dermatitis with satellite pustules. The associated vaginitis may result in a thick white or yellowish lumpy discharge. Secondary candidiasis may also develop on pre-existing skin disorders. In children with seborrhoeic der-

matitis, generalised erythematous patches which are sharply demarcated and red papular pustular scaly satellite lesions may develop.² In a child with napkin dermatitis, the mouth should always be checked for oral thrush. The provisional diagnosis of candidal infection depends on the clinical picture. A vulval or vaginal swab should be taken for Gram stain to look for pseudohyphae and budding cells. A swab should also be placed in transport medium and cultured for definitive confirmation.

Treatment of candidal infection of neonates requires attention to hygiene and the use of simple barrier creams. Local therapy with antifungal creams may be necessary, for example clotrimazole. Napkin eruptions may be less likely to relapse if oral therapy is also given to eliminate yeasts from the bowel.⁶⁷ In older girls specific therapy with local antifungal cream is usually required. It may be necessary to insert cream into the vagina. To prevent relapse, attention should be paid to adequate toileting and hygiene. Predisposing causes such as recent antibiotics, diabetes mellitus and other skin diseases should be excluded especially in cases of recurrent infection.

Scabies

Scabies affects all races and social classes world-wide. It is usually transmitted by close physical contact. Indirect spread by clothing or bedding is not thought to be so important.⁶⁸ Overcrowding which is common in the developing countries and is invariably associated with poverty and poor hygiene encourages the spread of scabies.⁶⁹ The presence of scabies in given populations depends upon the social environment.

Scabies is most common in children and young adults with an equal sex incidence. A Sheffield study showed that scabies was introduced into households by school children and teenagers.⁷⁰ The commonest sources of infection were friends and relatives outside the home. The high incidence of scabies reported in teenage girls may be due to contact with younger children in large families and hand holding.

Scabies is caused by the mite *Sarcoptes scabiei*. The mites survive for 24 to 36 hours in room conditions.⁷¹ Live mites can be found in dust samples collected in the homes of infected patients.⁷² Fomites such as clothing and blankets are thought not to play a part in the transmission of scabies.^{73,74}

Copulation occurs in a small burrow excavated by the female. The gravid female then enlarges the burrow and begins egg laying. She lays two to three eggs daily which hatch into larvae in three to four days. These migrate to the surface, burrow again and moult, producing nymphs before becoming adults about ten days after hatching.⁶⁹

The symptomatology induced by this infestation may be the result of sensitisation of the host to the mite and its products.⁷⁵ The most obvious manifestation of scabies is itching,⁶⁹ occurring 3–4 weeks after infection is

acquired. The nocturnal character of the itch supports the view that the itch occurs only when the mite is burrowing⁷⁶ rather than as an allergic phenomenon.⁷⁷ In older children and adolescents the clinical pattern is similar to that seen in adults; preferred sites are interdigital spaces, wrists, elbows, feet, buttocks and axillae. In young children, vesicles rather than tunnels are often the rule.⁷⁸⁻⁷⁹ The palms, soles, face and scalp may be affected without the presence of burrows. Red/brown nodules most often found in the axillae, groins and genitalia are a less common variant.

Scabies is usually diagnosed clinically and confirmed by taking scrapings from a burrow with a blunt instrument, placing the material in a drop of 10% KOH and examining the slide with a microscope to identify the mite.⁸⁰

Secondary features may confuse the clinical picture. Eczematous changes which can be widespread and severe in children may mimic atopic dermatitis and seborrhoeic dermatitis.⁷⁸⁻⁸⁰ The inappropriate use of topical steroids will modify the clinical picture in these cases. Papular lesions can be confused with papular urticaria, chicken pox, drug eruptions or dermatitis herpetiformis. Secondary infection may manifest itself as folliculitis impetigo, obscuring the primary diagnosis of scabies.⁸¹

Several drugs are available for treatment of scabies in adults. These include malathion, lindane, crotamiton, benzyl benzoate, and the new treatment permethrin. In children there is greater potential for toxicity because of a larger body surface area to mass ratio which increases the potential for percutaneous absorption. Benzyl benzoate is a recognised dermal irritant requiring several applications to ensure successful treatment and should be avoided in children. Similarly, lindane is potentially neurotoxic and should be avoided. Aqueous malathion 0.5% lotion has been successfully used with a single application,⁸¹ and can be used on the face and scalp if care is taken to avoid the eyes and mouth. Permethrin 5% cream has been found to be as effective as lindane and more effective than crotamiton in treating scabies.⁸²⁻⁸³ Animal studies indicate that permethrin is one third less toxic than malathion and one fortieth as toxic as lindane. The cream is applied from the neck down in adults but in infants and toddlers the cream can be applied to the face, neck and scalp. The cream should be left on for 8–14 hours then washed off. In children aged two months to five years 89% were clear of all lesions at one month with permethrin cream compared with 60% with crotamiton. Oral anti-histamines may occasionally be required to relieve the itching.

Pubic lice

Phthirus pubis, the pubic louse has been known for centuries. It is a broad, short, blood sucking insect with a pincer adaptation on the first pair of legs for gripping hair. It anchors itself to a hair on either side with the second and third pair of legs. It therefore prefers pubic and axillary hair because these

are far enough apart to suit the span of louse (2 mm) whereas the scalp hairs are too close together. It tends to remain attached to one place for hours or days, feeding intermittently by injecting saliva through its mouth parts. The average life of an adult is 17–22 days with an incubation period for eggs of 7–8 days and the life cycle from egg to adult is 22–27 days.⁶⁹

Louse infestation is widespread and its incidence fluctuates over long periods. It is spread by close contact, usually sexual intercourse. Other modes of transmission in children include bed sharing with an infested mother or siblings. Close contact with other child carers (for example, nurses) has been reported to cause infection.⁸⁴ Spread may also occasionally occur through contact on a toilet seat with egg infested pubic hairs which have been loosened by scratching.⁸⁵

Usually the only complaint of infection is itching, which occurs in the infested areas of the body. In adults the pubic and axillary hairs are most often affected, but the lice may spread to other areas of the body with heavy infestation. In children without pubic or axillary hair the eyelashes and eyebrows can be affected. Involvement of the scalp is very rare and occurs in children only in the presence of eyelash involvement.⁶⁹⁻⁸⁶

The eyelid infestation is seen as a marked yellowish encrustation. The crusts consist of lice together with eggs and reddish-brown faecal deposits.⁸⁷ There is often accompanying oedema of the eyelid. The eggs can be seen attached to the eyelashes as shiny greyish oval globules.

The treatment of pubic lice in children is simple. The safest preparations are 1% permethrin and 0.5% malathion.⁸⁰⁻⁸³ Infestation of the eyelid is treated with twice-daily applications of petrolcin for 7–10 days. Alternative treatments include anticholinergic eye ointments (0.75% phyostigmine ophthalmic⁸⁸⁻⁸⁹) or yellow oxide of mercury ("Golden eye ointment"). Clothing and bed linen must be washed and heat dried after treatment. Contacts should be treated.

Future developments

With our present knowledge of vaginal flora in children, uncertainty persists as to which organisms are normally present, which organisms are pathogenic and which are predominantly acquired by sexual activity and may therefore be markers of abuse in young girls.

Further information is required on the prevalence of vaginal organisms in children. Selection of satisfactory control groups for these studies will be problematic. There is an inherent difficulty in identifying children with undisclosed abuse or sexual activity. Studies to date have not assessed antibiotic use in the abused or control population and there is no information regarding the re-establishment of vaginal flora after antibiotic use. Another factor which has not been addressed is the relationship of the examination of the child to the time of abuse. This has not been consistently reported in any study of child abuse and infection. It may be that the transmission of some

organisms is not direct, but by opportunistic colonisation related to factors associated with sexual activity. These factors need elucidation.

Further well controlled and multi-centre studies should be conducted which take account of age, maturity and race of the children under investigation. Meanwhile the presence of mycoplasmas, bacterial vaginosis (including isolation of *G vaginalis*), should alert clinicians to look for indicators of possible abuse. It should also be remembered that scabies and pubic lice are not commonly sexually transmitted in children.

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